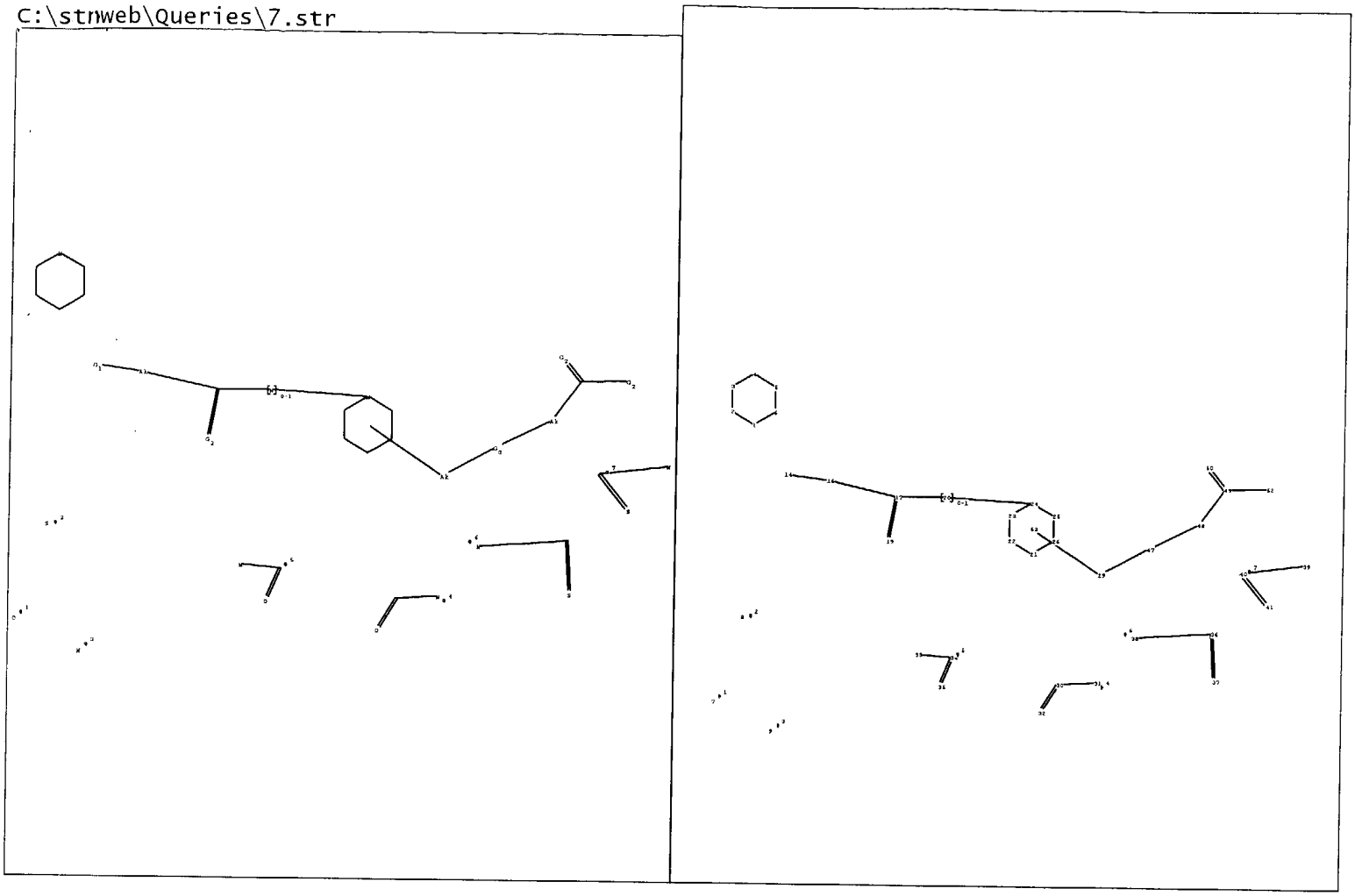
C:\stnweb\Queries\7.str



chain nodes :
7 8 9 14 16 17 19 20 29 30 31 32 33 34 35 36 37 38 39 40 41 47 48
49 50 52
ring nodes :
1 2 3 4 5 6 21 22 23 24 25 26
chain bonds :
14-16 16-17 17-19 17-20 20-24 29-47 30-31 30-32 33-34 34-35 36-37 36-38 39-40
40-41 47-48 48-49 49-52 49-50
ring bonds :
1-2 1-6 2-3 3-4 4-5 5-6 21-22 21-26 22-23 23-24 24-25 25-26
exact/norm bonds :
1-2 1-6 2-3 3-4 4-5 5-6 14-16 16-17 17-19 17-20 20-24 21-22 21-26 22-23
23-24 24-25 25-26 29-47 30-31 30-32 33-34 34-35 36-37 36-38 39-40 40-41 47-48
48-49 49-52 49-50
isolated ring systems :
containing 1 : 21 :

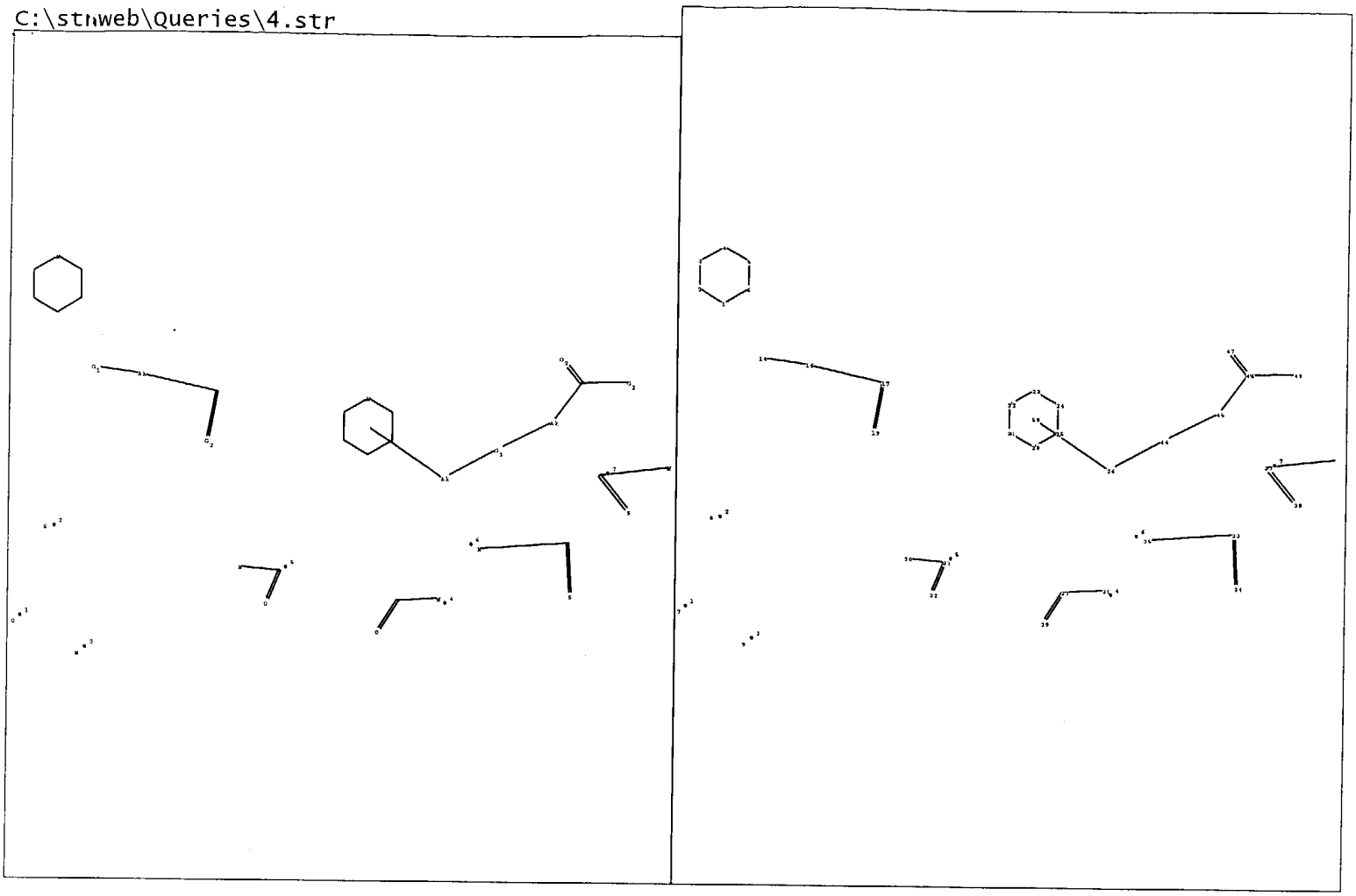
G1:[*1],[*2],[*3]

G2:O,S

G3:[*4],[*5],[*6],[*7]

Match level :
1:Atom 2:Atom 3:Atom 4:Atom 5:Atom 6:Atom 7:CLASS 8:CLASS 9:CLASS 14:CLASS
16:CLASS 17:CLASS 19:CLASS 20:CLASS 21:Atom 22:Atom 23:Atom 24:Atom 25:Atom
26:Atom 29:CLASS 30:CLASS 31:CLASS 32:CLASS 33:CLASS 34:CLASS 35:CLASS 36:CLASS
37:CLASS 38:CLASS 39:CLASS 40:CLASS 41:CLASS 47:CLASS 48:CLASS 49:CLASS 50:CLASS
52:CLASS 53:CLASS

C:\stnweb\Queries\4.str



chain nodes :
7 8 9 14 16 17 19 26 27 28 29 30 31 32 33 34 35 36 37 38 44 45 46
47 49
ring nodes :
1 2 3 4 5 6 20 21 22 23 24 25
chain bonds :
14-16 16-17 17-19 26-44 27-28 27-29 30-31 31-32 33-34 33-35 36-37 37-38 44-45
45-46 46-49 46-47
ring bonds :
1-2 1-6 2-3 3-4 4-5 5-6 20-21 20-25 21-22 22-23 23-24 24-25
exact/norm bonds :
1-2 1-6 2-3 3-4 4-5 5-6 14-16 16-17 17-19 20-21 20-25 21-22 22-23 23-24
24-25 26-44 27-28 27-29 30-31 31-32 33-34 33-35 36-37 37-38 44-45 45-46 46-49
46-47
isolated ring systems :
containing 1 : 20 :

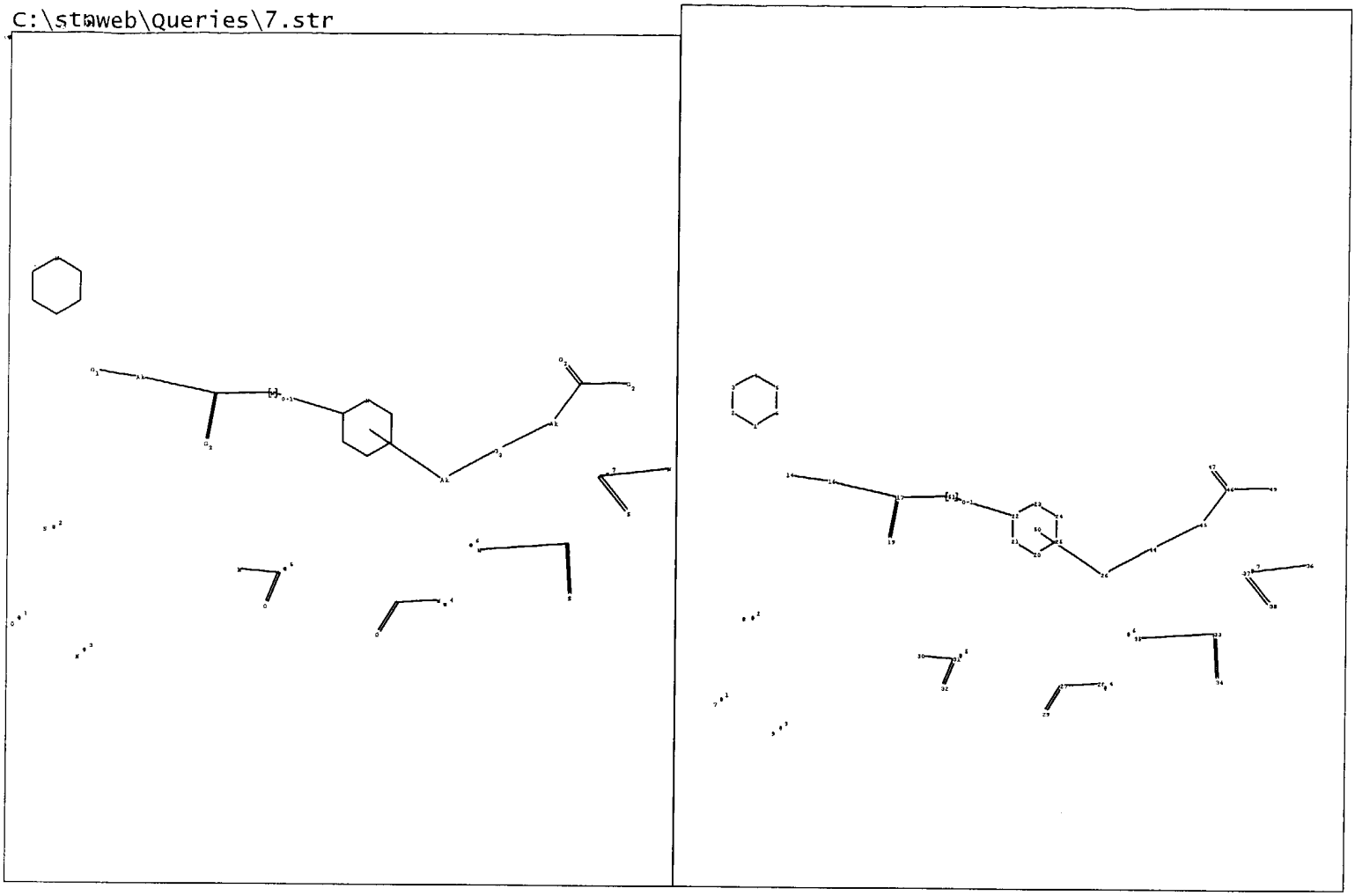
G1:[*1],[*2],[*3]

G2:O,S

G3:[*4],[*5],[*6],[*7]

Match level :
1:Atom 2:Atom 3:Atom 4:Atom 5:Atom 6:Atom 7:CLASS 8:CLASS 9:CLASS 14:CLASS
16:CLASS 17:CLASS 19:CLASS 20:Atom 21:Atom 22:Atom 23:Atom 24:Atom 25:Atom
26:CLASS 27:CLASS 28:CLASS 29:CLASS 30:CLASS 31:CLASS 32:CLASS 33:CLASS 34:CLASS
35:CLASS 36:CLASS 37:CLASS 38:CLASS 44:CLASS 45:CLASS 46:CLASS 47:CLASS 49:CLASS
50:CLASS

C:\stnweb\Queries\7.str



chain nodes :
7 8 9 14 16 17 19 26 27 28 29 30 31 32 33 34 35 36 37 38 44 45 46
47 49 51
ring nodes :
1 2 3 4 5 6 20 21 22 23 24 25
chain bonds :
14-16 16-17 17-19 17-51 22-51 26-44 27-28 27-29 30-31 31-32 33-34 33-35 36-37
37-38 44-45 45-46 46-49 46-47
ring bonds :
1-2 1-6 2-3 3-4 4-5 5-6 20-21 20-25 21-22 22-23 23-24 24-25
exact/norm bonds :
1-2 1-6 2-3 3-4 4-5 5-6 14-16 16-17 17-19 17-51 20-21 20-25 21-22 22-23
22-51 23-24 24-25 26-44 27-28 27-29 30-31 31-32 33-34 33-35 36-37 37-38 44-45
45-46 46-49 46-47
isolated ring systems :
containing 1 : 20 :

G1:[*1],[*2],[*3]

G2:O,S

G3:[*4],[*5],[*6],[*7]

Match level :
1:Atom 2:Atom 3:Atom 4:Atom 5:Atom 6:Atom 7:CLASS 8:CLASS 9:CLASS 14:CLASS
16:CLASS 17:CLASS 19:CLASS 20:Atom 21:Atom 22:Atom 23:Atom 24:Atom 25:Atom
26:CLASS 27:CLASS 28:CLASS 29:CLASS 30:CLASS 31:CLASS 32:CLASS 33:CLASS 34:CLASS
35:CLASS 36:CLASS 37:CLASS 38:CLASS 44:CLASS 45:CLASS 46:CLASS 47:CLASS 49:CLASS
50:CLASS 51:CLASS

```
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NEWS  1              Web Page URLs for STN Seminar Schedule - N. America
NEWS  2              "Ask CAS" for self-help around the clock
NEWS  3   May 10     PROUSDDR now available on STN
NEWS  4   May 19     PROUSDDR: One FREE connect hour, per account, in both May
                     and June 2004
NEWS  5   May 12     EXTEND option available in structure searching
NEWS  6   May 12     Polymer links for the POLYLINK command completed in REGISTRY
NEWS  7   May 17     FRFULL now available on STN
NEWS  8   May 27     New UPM (Update Code Maximum) field for more efficient patent
                     SDIs in CAplus
NEWS  9   May 27     CAplus super roles and document types searchable in REGISTRY
NEWS 10   May 27     Explore APOLLIT with free connect time in June 2004
NEWS 11   Jun 22     STN Patent Forums to be held July 19-22, 2004
NEWS 12   Jun 28     Additional enzyme-catalyzed reactions added to CASREACT
NEWS 13   Jun 28     ANTE, AQUALINE, BIOENG, CIVILENG, ENVIROENG, MECHENG,
                     and WATER from CSA now available on STN(R)

NEWS EXPRESS  MARCH 31 CURRENT WINDOWS VERSION IS V7.00A, CURRENT
              MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
              AND CURRENT DISCOVER FILE IS DATED 26 APRIL 2004
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NEWS LOGIN    Welcome Banner and News Items
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FILE 'HOME' ENTERED AT 11:25:36 ON 01 JUL 2004

=> file reg
COST IN U.S. DOLLARS                                  SINCE FILE      TOTAL
                                                        ENTRY     SESSION
FULL ESTIMATED COST                                      0.21        0.21

FILE 'REGISTRY' ENTERED AT 11:25:47 ON 01 JUL 2004
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provided by InfoChem.

STRUCTURE FILE UPDATES:   30 JUN 2004  HIGHEST RN 701907-96-2
DICTIONARY FILE UPDATES:  30 JUN 2004  HIGHEST RN 701907-96-2

TSCA INFORMATION NOW CURRENT THROUGH JANUARY 6, 2004

  Please note that search-term pricing does apply when
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   conducting SmartSELECT searches.

 Crossover limits have been increased.  See HELP CROSSOVER for details.

 Experimental and calculated property data are now available. For more
 information enter HELP PROP at an arrow prompt in the file or refer
 to the file summary sheet on the web at:
 http://www.cas.org/ONLINE/DBSS/registryss.html

 =>
 L1       STRUCTURE UPLOADED

 => d l1
 L1 HAS NO ANSWERS
 L1               STR

 => s l1
 SAMPLE SEARCH INITIATED 11:32:09 FILE 'REGISTRY'
 SAMPLE SCREEN SEARCH COMPLETED -  18108 TO ITERATE

   5.5% PROCESSED     1000 ITERATIONS                                    0 ANSWERS
 INCOMPLETE SEARCH (SYSTEM LIMIT EXCEEDED)
 SEARCH TIME: 00.00.01

 FULL FILE PROJECTIONS:  ONLINE  **COMPLETE**
                         BATCH   **COMPLETE**
 PROJECTED ITERATIONS:       354108 TO   370212
 PROJECTED ANSWERS:               0 TO        0

 L2             0 SEA SSS SAM L1

 => s l1 full
 THE ESTIMATED SEARCH COST FOR FILE 'REGISTRY' IS 155.00 U.S. DOLLARS
 DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N or END:y
 FULL SEARCH INITIATED 11:32:14 FILE 'REGISTRY'
 FULL SCREEN SEARCH COMPLETED - 359409 TO ITERATE

 100.0% PROCESSED  359409 ITERATIONS                                   0 ANSWERS
 SEARCH TIME: 00.00.10

 L3             0 SEA SSS FUL L1

 =>
 L4       STRUCTURE UPLOADED

 => d l4
 L4 HAS NO ANSWERS
 L4               STR

 => s l4
 SAMPLE SEARCH INITIATED 11:33:17 FILE 'REGISTRY'
 SAMPLE SCREEN SEARCH COMPLETED -  24405 TO ITERATE

   4.1% PROCESSED     1000 ITERATIONS                                    0 ANSWERS
 INCOMPLETE SEARCH (SYSTEM LIMIT EXCEEDED)
 SEARCH TIME: 00.00.01

 FULL FILE PROJECTIONS:  ONLINE  **INCOMPLETE**
                         BATCH   **COMPLETE**
 PROJECTED ITERATIONS:       478761 TO   497439
```

```
PROJECTED ANSWERS:                0 TO        0

L5             0 SEA SSS SAM L4

=>
L6       STRUCTURE UPLOADED

=> d l6
L6 HAS NO ANSWERS
L6                STR

=> s l6
SAMPLE SEARCH INITIATED 11:34:52 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED -  14259 TO ITERATE

  7.0% PROCESSED    1000 ITERATIONS                                0 ANSWERS
INCOMPLETE SEARCH (SYSTEM LIMIT EXCEEDED)
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS:  ONLINE  **COMPLETE**
                        BATCH   **COMPLETE**
PROJECTED ITERATIONS:       278031 TO   292329
PROJECTED ANSWERS:               0 TO        0

L7             0 SEA SSS SAM L6

=> s l6 full
THE ESTIMATED SEARCH COST FOR FILE 'REGISTRY' IS 155.00 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N or END:y
FULL SEARCH INITIATED 11:34:56 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED - 283506 TO ITERATE

100.0% PROCESSED  283506 ITERATIONS                              0 ANSWERS
SEARCH TIME: 00.00.09

L8             0 SEA SSS FUL L6

=> file hcaplus
COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                 ENTRY    SESSION
FULL ESTIMATED COST                              316.72     316.93

FILE 'HCAPLUS' ENTERED AT 11:35:36 ON 01 JUL 2004
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FILE COVERS 1907 - 1 Jul 2004  VOL 141 ISS 1
FILE LAST UPDATED: 30 Jun 2004  (20040630/ED)
```

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 This file contains CAS Registry Numbers for easy and accurate
 substance identification.


=> s glycoprotein IIb/IIIa () antagonist?
'IIIA' IS NOT A VALID FIELD CODE
             0 GLYCOPROTEIN IIB/IIIA
        208653 ANTAGONIST?
L9           0 GLYCOPROTEIN IIB/IIIA (W) ANTAGONIST?

=> s glycoprotein? () antagonist?
        135487 GLYCOPROTEIN?
        208653 ANTAGONIST?
L10         75 GLYCOPROTEIN? (W) ANTAGONIST?

=> s l10 and thrombus () formation
          7244 THROMBUS
             1 THROMBUSES
          2294 THROMBI
            16 THROMBIS
          8475 THROMBUS
                 (THROMBUS OR THROMBUSES OR THROMBI OR THROMBIS)
       2471824 FORMATION
         51288 FORMATIONS
       2500775 FORMATION
                 (FORMATION OR FORMATIONS)
          3076 THROMBUS (W) FORMATION
L11          2 L10 AND THROMBUS (W) FORMATION

=> s l11 and review/dt
       1738650 REVIEW/DT
L12          1 L11 AND REVIEW/DT

=> d l12, ibib abs, 1

L12  ANSWER 1 OF 1  HCAPLUS  COPYRIGHT 2004 ACS on STN
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Full Text | Citing References

```
ACCESSION NUMBER:         2003:328730  HCAPLUS
DOCUMENT NUMBER:          139:78262
TITLE:                    Platelet glycoprotein IIb/IIIa antagonists: their
                          interaction with low-molecular-weight heparins and
                          direct thrombin inhibitors
AUTHOR(S):                Bapat, Amol S.; Yazbek, Naji; Kleiman, Neal S.
CORPORATE SOURCE:         The Methodist DeBakey Heart Center, Baylor College of
                          Medicine, Houston, TX, USA
SOURCE:                   Platelet Glycoprotein IIb/IIIa Inhibitors in
                          Cardiovascular Disease (2nd Edition) (2003), 341-364.
                          Editor(s): Lincoff, A. Michael.  Humana Press Inc.:
                          Totowa, N. J.
                          CODEN: 69DVLO; ISBN: 1-58829-185-5
DOCUMENT TYPE:            Conference; General Review
LANGUAGE:                 English
AB    A review.  Thrombin's central role in platelet activation, thrombus
      formation, and propagation is abundantly clear from numerous animal and
      human studies.  Although unfractionated heparin has been instrumental in
      reducing events in many cardiac settings, its practical and theor.
      limitations have become more evident.  Low-mol.-wt. heparins and direct
      thrombin inhibitors, which have theor. and practical advantages over
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     unfractionated heparin, have shown favorable outcomes when used in a
     variety of cardiac settings.  In combination with a GPIIb/IIIa antagonist,
     preliminary data have provided evidence of safety and efficacy.  New
     randomized clin. trials are under way to det. whether combination therapy
     may be the next std. of care in modem practice.
REFERENCE COUNT:          105   THERE ARE 105 CITED REFERENCES AVAILABLE FOR
                                THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE
                                FORMAT


=> d his

     (FILE 'HOME' ENTERED AT 11:25:36 ON 01 JUL 2004)

     FILE 'REGISTRY' ENTERED AT 11:25:47 ON 01 JUL 2004
L1              STRUCTURE UPLOADED
L2            0 S L1
L3            0 S L1 FULL
L4              STRUCTURE UPLOADED
L5            0 S L4
L6              STRUCTURE UPLOADED
L7            0 S L6
L8            0 S L6 FULL

     FILE 'HCAPLUS' ENTERED AT 11:35:36 ON 01 JUL 2004
L9            0 S GLYCOPROTEIN IIB/IIIA () ANTAGONIST?
L10          75 S GLYCOPROTEIN? () ANTAGONIST?
L11           2 S L10 AND THROMBUS () FORMATION
L12           1 S L11 AND REVIEW/DT

=> s l10 and arterial? () thrombosis?
         79957 ARTERIAL?
         18393 THROMBOSIS?
          1182 ARTERIAL? (W) THROMBOSIS?
L13          1 L10 AND ARTERIAL? (W) THROMBOSIS?

=> s l13 and review/dt
       1738650 REVIEW/DT
L14           0 L13 AND REVIEW/DT

=> s l10 and arterial? () sclerosis?
         79957 ARTERIAL?
         18569 SCLEROSIS?
            36 ARTERIAL? (W) SCLEROSIS?
L15          0 L10 AND ARTERIAL? (W) SCLEROSIS?

=> s l10 and ischemic? () heart? () disease?
         37886 ISCHEMIC?
           149 ISCHEM
           149 ISCHEM
                 (ISCHEM)
         37939 ISCHEMIC?
                 (ISCHEMIC? OR ISCHEM)
        312570 HEART?
        785618 DISEASE?
          3471 ISCHEMIC? (W) HEART? (W) DISEASE?
L16          0 L10 AND ISCHEMIC? (W) HEART? (W) DISEASE?

=> s l10 and ischem? () heart?
         66845 ISCHEM?
```

```
         312570 HEART?
           6315 ISCHEM? (W) HEART?
L17           0 L10 AND ISCHEM? (W) HEART?

=> s l10 and angina? () pectoris?
           7629 ANGINA?
           5768 PECTORIS?
           5607 ANGINA? (W) PECTORIS?
L18           4 L10 AND ANGINA? (W) PECTORIS?

=> s l18 and review/dt
        1738650 REVIEW/DT
L19           2 L18 AND REVIEW/DT

=> d l19, ibib abs, 1-2

L19  ANSWER 1 OF 2  HCAPLUS  COPYRIGHT 2004 ACS on STN
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Full Text | Citing References

ACCESSION NUMBER: 2001:688051 HCAPLUS
DOCUMENT NUMBER: 136:79085
TITLE: Oral Glycoprotein IIb/IIIa Antagonists for Unstable Angina - Is There Still a Chance for the Oral Substances?
AUTHOR(S): Darius, H.
CORPORATE SOURCE: Department of Medicine II, Johannes Gutenberg-University, Mainz, Germany
SOURCE: Thrombosis Research (2001), 103(Suppl. 1), S117-S124
CODEN: THBRAA; ISSN: 0049-3848
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. The i.v. glycoprotein IIb/IIIa antagonists abciximab, tirofiban and eptifibatide are well accepted for the therapy of patients with unstable angina and/or as concomitant medication during coronary interventions. Despite the fact that these drugs are not used in all patients presenting with unstable angina during coronary interventions, the scientific evidence is overwhelming including the substantial redn. in mortality 3 yr after utilization of abciximab for coronary interventions in patients with unstable angina. In addn. to these two indications, i.v. glycoprotein IIb/IIIa antagonists are currently being investigated for use in patients undergoing carotid artery interventions, peripheral arterial interventions and stroke, as well as adjunct therapy in patients undergoing fibrinolytic therapy during acute myocardial infarction. In contrast, the large trials being performed in patients with unstable angina and following coronary interventions using oral glycoprotein IIb/IIIa antagonists have been very disappointing. There were only minor therapeutic effects detectable, resulting in a slight redn. in ischemic cardiac events in some investigations, however, in all studies, there was a slight trend towards an increased mortality in the glycoprotein IIb/IIIa receptor-antagonist-treated group of patients. In meta-anal., an approx. 35% relative increase in mortality has been calcd. for patients being treated long term with the oral **glycoprotein antagonists**. The reason for this therapeutic failure is still unknown, however, the limited bioavailability of these drugs, together with our still very limited knowledge about the regulation of the platelet fibrinogen receptor, may be partially responsible for this therapeutic failure. Other compds. with improved pharmacokinetic properties are currently in clin. development.

REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2000:86547 HCAPLUS
DOCUMENT NUMBER: 132:216396
TITLE: Tirofiban (Aggrastat)
AUTHOR(S): Cook, Jacquelynn J.; Bednar, Bohumil; Lynch, Joseph J., Jr.; Gould, Robert J.; Egbertson, Melissa S.; Halczenko, Wasyl; Duggan, Mark E.; Hartman, George D.; Lo, Man-Wai; Murphy, Gail M.; Deckelbaum, Lawrence I.; Sax, Frederick L.; Barr, Eliav
CORPORATE SOURCE: Merck Research Laboratories, West Point, PA, 19486, USA
SOURCE: Cardiovascular Drug Reviews (1999), 17(3), 199-224
CODEN: CDREEA; ISSN: 0897-5957
PUBLISHER: Neva Press
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review with 61 refs. is given on chem., in vitro and in vivo preclin. and clin. pharmacol. of tirofiban. Tirofiban is a specific antagonist of the platelet surface glycoprotein (GP) IIb/IIIa receptor, the final common pathway for platelet aggregation. The phase III clin. program demonstrated that therapy with tirofiban, in combination with heparin, resulted in a substantial and durable (up to 6 mo) redn. in the incidence of adverse cardiovascular sequelae in patients presenting with unstable **angina pectoris**/non-Q-wave myocardial infarction. The obsd. benefit of tirofiban included a redn. in the incidence of irreversible myocardial damage as well as reversible cardiovascular morbid events. The benefit of therapy with tirofiban plus heparin was obsd. regardless of the revascularization strategy used in the in-hospital management of the patients. In patients with acute coronary syndromes in whom tirofiban (with heparin and aspirin) was initiated in the setting of percutaneous coronary angioplasty or arterectomy, tirofiban reduced acute cardiac ischemic complications related to abrupt closure of the treated coronary vessel during the early part of the 30-d-follow-up period. Therapy with tirofiban, heparin, and aspirin was accompanied not only by a beneficial clin. efficacy profile but also by an acceptable safety profile. Therapy with tirofiban was in general well tolerated, with only small increases in major bleeding events and low incidence of thrombocytopenia. The resultant modest increase in bleeding events and transfusion requirements stands in contrast to the redn. of irreversible cardiovascular sequelae of death/myocardial infarction.

REFERENCE COUNT: 61 THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

```
=> s l10 and myocardial? () infarct?
         54567 MYOCARDIAL?
         29730 INFARCT?
         17598 MYOCARDIAL? (W) INFARCT?
L20         10 L10 AND MYOCARDIAL? (W) INFARCT?

=> s l20 and review/dt
       1738650 REVIEW/DT
L21          8 L20 AND REVIEW/DT

=> d l21, ibib abs, 1-8

L21 ANSWER 1 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN
```

Full Text | Citing References

ACCESSION NUMBER: 2003:744618 HCAPLUS
DOCUMENT NUMBER: 140:331467
TITLE: Development and use of platelet **glycoprotein antagonists** in heart disease
AUTHOR(S): Bennett, Joel S.
CORPORATE SOURCE: Hematology-Oncology Division, Department of Medicine, University of Pennsylvania School of Medicine, Philadelphia, PA, USA
SOURCE: Cardiac Drug Development Guide (2003), 107-122. Editor(s): Pugsley, Michael K. Humana Press Inc.: Totowa, N. J.
CODEN: 69ENK4; ISBN: 1-58829-097-2
DOCUMENT TYPE: Conference; **General Review**
LANGUAGE: English
AB A review on platelet **glycoprotein antagonists**, particularly the GPIIb/IIIa antagonists. Ligand binding to GPIIb/IIIa on activated platelets is a prerequisite for platelet aggregation, and for the formation of arterial thrombi. Thus, GPIIb/IIIa has been a major target for antiplatelet drug development. Three i.v. GPIIb/IIIa antagonists (abciximab, eptifibatide, and tirofiban) have been shown in large multicenter clin. trials to be effective in reducing the incidence of a common endpoint of death, **myocardial infarction**, and urgent coronary artery revascularization in patients with acute coronary syndromes.
REFERENCE COUNT: 109 THERE ARE 109 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 2 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:328730 HCAPLUS
DOCUMENT NUMBER: 139:78262
TITLE: Platelet glycoprotein IIb/IIIa antagonists: their interaction with low-molecular-weight heparins and direct thrombin inhibitors
AUTHOR(S): Bapat, Amol S.; Yazbek, Naji; Kleiman, Neal S.
CORPORATE SOURCE: The Methodist DeBakey Heart Center, Baylor College of Medicine, Houston, TX, USA
SOURCE: Platelet Glycoprotein IIb/IIIa Inhibitors in Cardiovascular Disease (2nd Edition) (2003), 341-364. Editor(s): Lincoff, A. Michael. Humana Press Inc.: Totowa, N. J.
CODEN: 69DVLO; ISBN: 1-58829-185-5
DOCUMENT TYPE: Conference; **General Review**
LANGUAGE: English
AB A review. Thrombin's central role in platelet activation, thrombus formation, and propagation is abundantly clear from numerous animal and human studies. Although unfractionated heparin has been instrumental in reducing events in many cardiac settings, its practical and theor. limitations have become more evident. Low-mol.-wt. heparins and direct thrombin inhibitors, which have theor. and practical advantages over unfractionated heparin, have shown favorable outcomes when used in a variety of cardiac settings. In combination with a GPIIb/IIIa antagonist, preliminary data have provided evidence of safety and efficacy. New randomized clin. trials are under way to det. whether combination therapy may be the next std. of care in modem practice.
REFERENCE COUNT: 105 THERE ARE 105 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE

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L21 ANSWER 3 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:215934 HCAPLUS
DOCUMENT NUMBER: 138:352321
TITLE: Glycoprotein IIb/IIIa inhibition in early intent-to-stent treatment of acute coronary syndromes: EPISTENT, ADMIRAL, CADILLAC, and TARGET
AUTHOR(S): Moliterno, David J.; Chan, Albert W.
CORPORATE SOURCE: Department of Cardiovascular Medicine, The Cleveland Clinic Foundation, Cleveland, OH, USA
SOURCE: Journal of the American College of Cardiology (2003), 41(4, Suppl.), 49S-54S
CODEN: JACCDI; ISSN: 0735-1097
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review. The acute coronary syndromes (ACS), with or without ST-segment elevation, share a common pathophysiol. of activated platelets and thrombin generation stimulated by plaque erosion and rupture. Both mech. and pharmacol. treatment strategies have evolved in an attempt to improve reperfusion at the myocardial tissue level. Intracoronary stents have lowered the incidence of abrupt vessel closure and restenosis, while potent platelet inhibition from i.v. glycoprotein IIb/IIIa antagonists has reduced the rate of periprocedural **myocardial infarction** and late mortality. Abciximab has well-established clin. benefits in percutaneous revascularization trials, and several recent landmark studies have evaluated the efficacy of concomitant abciximab during mech. reperfusion therapy in the setting of ACS. These trials are reviewed, and an overall perspective is provided.
REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 4 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:688051 HCAPLUS
DOCUMENT NUMBER: 136:79085
TITLE: Oral Glycoprotein IIb/IIIa Antagonists for Unstable Angina - Is There Still a Chance for the Oral Substances?
AUTHOR(S): Darius, H.
CORPORATE SOURCE: Department of Medicine II, Johannes Gutenberg-University, Mainz, Germany
SOURCE: Thrombosis Research (2001), 103(Suppl. 1), S117-S124
CODEN: THBRAA; ISSN: 0049-3848
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review. The i.v. glycoprotein IIb/IIIa antagonists abciximab, tirofiban and eptifibatide are well accepted for the therapy of patients with unstable angina and/or as concomitant medication during coronary interventions. Despite the fact that these drugs are not used in all patients presenting with unstable angina during coronary interventions, the scientific evidence is overwhelming including the substantial redn. in mortality 3 yr after utilization of abciximab for coronary interventions in patients with unstable angina. In addn. to these two indications, i.v. glycoprotein IIb/IIIa antagonists are currently being investigated for use

in patients undergoing carotid artery interventions, peripheral arterial interventions and stroke, as well as adjunct therapy in patients undergoing fibrinolytic therapy during acute **myocardial infarction**. In contrast, the large trials being performed in patients with unstable angina and following coronary interventions using oral glycoprotein IIb/IIIa antagonists have been very disappointing. There were only minor therapeutic effects detectable, resulting in a slight redn. in ischemic cardiac events in some investigations, however, in all studies, there was a slight trend towards an increased mortality in the glycoprotein IIb/IIIa receptor-antagonist-treated group of patients. In meta-anal., an approx. 35% relative increase in mortality has been calcd. for patients being treated long term with the oral **glycoprotein antagonists**. The reason for this therapeutic failure is still unknown, however, the limited bioavailability of these drugs, together with our still very limited knowledge about the regulation of the platelet fibrinogen receptor, may be partially responsible for this therapeutic failure. Other compds. with improved pharmacokinetic properties are currently in clin. development.

REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 5 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2000:86547 HCAPLUS
DOCUMENT NUMBER: 132:216396
TITLE: Tirofiban (Aggrastat)
AUTHOR(S): Cook, Jacquelynn J.; Bednar, Bohumil; Lynch, Joseph J., Jr.; Gould, Robert J.; Egbertson, Melissa S.; Halczenko, Wasyl; Duggan, Mark E.; Hartman, George D.; Lo, Man-Wai; Murphy, Gail M.; Deckelbaum, Lawrence I.; Sax, Frederick L.; Barr, Eliav
CORPORATE SOURCE: Merck Research Laboratories, West Point, PA, 19486, USA
SOURCE: Cardiovascular Drug Reviews (1999), 17(3), 199-224
CODEN: CDREEA; ISSN: 0897-5957
PUBLISHER: Neva Press
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review with 61 refs. is given on chem., in vitro and in vivo preclin. and clin. pharmacol. of tirofiban. Tirofiban is a specific antagonist of the platelet surface glycoprotein (GP) IIb/IIIa receptor, the final common pathway for platelet aggregation. The phase III clin. program demonstrated that therapy with tirofiban, in combination with heparin, resulted in a substantial and durable (up to 6 mo) redn. in the incidence of adverse cardiovascular sequelae in patients presenting with unstable angina pectoris/non-Q-wave **myocardial infarction**. The obsd. benefit of tirofiban included a redn. in the incidence of irreversible myocardial damage as well as reversible cardiovascular morbid events. The benefit of therapy with tirofiban plus heparin was obsd. regardless of the revascularization strategy used in the in-hospital management of the patients. In patients with acute coronary syndromes in whom tirofiban (with heparin and aspirin) was initiated in the setting of percutaneous coronary angioplasty or arterectomy, tirofiban reduced acute cardiac ischemic complications related to abrupt closure of the treated coronary vessel during the early part of the 30-d-follow-up period. Therapy with tirofiban, heparin, and aspirin was accompanied not only by a beneficial clin. efficacy profile but also by an acceptable safety profile. Therapy with tirofiban was in general well tolerated, with only small increases in major bleeding events and low incidence of thrombocytopenia. The resultant modest increase in bleeding events and transfusion requirements

stands in contrast to the redn. of irreversible cardiovascular sequelae of death/**myocardial infarction**.
REFERENCE COUNT: 61 THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 6 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN
Full Text | Citing References
ACCESSION NUMBER: 2000:54613 HCAPLUS
DOCUMENT NUMBER: 132:73123
TITLE: Glycoprotein IIB/IIIA antagonists: do they have a role in the management of unstable angina?
AUTHOR(S): Redwood, S.; Marber, M.; Jackson, G.
CORPORATE SOURCE: Cardiothoracic Unit, St Thomas' Hospital, London, E1 7EH, UK
SOURCE: International Journal of Clinical Practice (1999), 53(8), 618-622
CODEN: IJCPF9; ISSN: 1368-5031
PUBLISHER: Medicom International
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review with 36 refs. Plaque rupture, platelet aggregation and thrombosis have central roles in the pathogenesis of acute coronary syndromes. Despite several trials showing the benefit of aspirin and heparin in patients presenting with unstable angina and acute **myocardial infarction**, these patients are still at risk. This has prompted the development and evaluation of several new therapeutic agents including low mol. wt. heparin, new antiplatelet drugs (e.g. ticlopidine and clopidogrel), direct thrombin inhibitors, and i.v. and oral glycoprotein IIb/IIIa antagonists. The IIb/IIIa receptor is the "final common pathway" involved in platelet aggregation. Thus, whatever the stimulus for platelet activation, subsequent aggregation is mediated by the IIb/IIIa receptor binding fibrinogen. A variety of antibody, peptide and non-peptide compds. that block the IIb/IIIa receptor have been developed, and several studies have investigated the role of these agents in patients with acute coronary syndromes both within and outside the setting of percutaneous intervention. This article summarizes the studies to date using IIb/IIIa antagonists, and discusses their role in patients with non-ST segment elevation acute coronary syndromes.
REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 7 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN
Full Text | Citing References
ACCESSION NUMBER: 1999:32554 HCAPLUS
DOCUMENT NUMBER: 130:75678
TITLE: Glycoprotein IIb/IIIa-antagonists
AUTHOR(S): Darius, Harald; Grosser, T.
CORPORATE SOURCE: II. Medizinische Klinik Poliklinik, Johannes-Gutenberg-Universitaet, Mainz, D-55101, Germany
SOURCE: Haemostaseologie (Stuttgart) (1998), 18(4), 171-179
CODEN: HAEMD2; ISSN: 0720-9355
PUBLISHER: F. K. Schattauer Verlagsgesellschaft mbH
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: German
AB A review with 35 refs. is given on glycoprotein IIb/IIIa-antagonists. Due to advances in the functional and mol. characterization of the platelet fibrinogen receptor (glycoprotein IIb/IIIa receptor; integrin

αIIbβ3) the importance of fibrinogen binding for platelet aggregation became evident. Clin., this process is of major pathophysiol. relevance for the acute coronary syndrome. The 1st antagonist evaluated clin. was the blocking monoclonal antibody fragment abciximab which was tested with great success in coronary interventions and in patients with unstable angina. The incidence of cardiac adverse events was reduced by treatment with abciximab. There are synthetic antagonists available for i.v. application that were tested with success for the redn. of adverse event rates following coronary interventions and during unstable angina. The rate of bleeding complications, that was increased in the initial studies, was markedly reduced by diminution of the heparin dosing. Addnl. indications, like treatment of patients with acute **myocardial infarction** during reperfusion therapy are currently studied intensively. Another area of intense clin. research are orally available GPIIb/IIIa antagonists, which are being developed for the treatment of patients following coronary interventions and acute coronary syndromes.

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 8 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1998:616401 HCAPLUS
DOCUMENT NUMBER: 130:77
TITLE: Eptifibatide; platelet antiaggregatory, glycoprotein IIb/IIIa antagonist, fibrinogen receptor antagonist
AUTHOR(S): Scarborough, Robert M.
CORPORATE SOURCE: Dept. of Medicinal Chemistry, COR Therapeutics, Inc., South San Francisco, CA, 94080, USA
SOURCE: Drugs of the Future (1998), 23(6), 585-590
CODEN: DRFUD4; ISSN: 0377-8282
PUBLISHER: Prous Science
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review with 41 refs. is given on eptifibatide. Eptifibatide was developed as a short-acting parenteral antithrombotic agent to be used during percutaneous coronary interventions, for the treatment of unstable angina and as an adjunct to thrombolytic agents for the treatment of acute **myocardial infarction**. It is a highly specific cyclic arginyl-glycyl-aspartyl sequence-like heptapeptide antagonist of the platelet glycoprotein IIb/IIIa. Pharmacol. actions, pharmacokinetics, metab., toxicity, and clin. studies are described.

REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 11:25:36 ON 01 JUL 2004)

FILE 'REGISTRY' ENTERED AT 11:25:47 ON 01 JUL 2004
L1 STRUCTURE UPLOADED
L2 0 S L1
L3 0 S L1 FULL
L4 STRUCTURE UPLOADED
L5 0 S L4
L6 STRUCTURE UPLOADED
L7 0 S L6
L8 0 S L6 FULL

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     FILE 'HCAPLUS' ENTERED AT 11:35:36 ON 01 JUL 2004
L9             0 S GLYCOPROTEIN IIB/IIIA () ANTAGONIST?
L10           75 S GLYCOPROTEIN? () ANTAGONIST?
L11            2 S L10 AND THROMBUS () FORMATION
L12            1 S L11 AND REVIEW/DT
L13            1 S L10 AND ARTERIAL? () THROMBOSIS?
L14            0 S L13 AND REVIEW/DT
L15            0 S L10 AND ARTERIAL? () SCLEROSIS?
L16            0 S L10 AND ISCHEMIC? () HEART? () DISEASE?
L17            0 S L10 AND ISCHEM? () HEART?
L18            4 S L10 AND ANGINA? () PECTORIS?
L19            2 S L18 AND REVIEW/DT
L20           10 S L10 AND MYOCARDIAL? () INFARCT?
L21            8 S L20 AND REVIEW/DT

=> s l10 and coronary () thrombos?
          53755 CORONARY
            220 CORONARIES
          53821 CORONARY
                  (CORONARY OR CORONARIES)
          21416 THROMBOS?
            619 CORONARY (W) THROMBOS?
L22           1 L10 AND CORONARY (W) THROMBOS?

=> s l22 and review/dt
        1738650 REVIEW/DT
L23           0 L22 AND REVIEW/DT

=> s l10 and ischemic () brain? () disease?
          37830 ISCHEMIC
              7 ISCHEMICS
          37832 ISCHEMIC
                  (ISCHEMIC OR ISCHEMICS)
         467379 BRAIN?
         785618 DISEASE?
             33 ISCHEMIC (W) BRAIN? (W) DISEASE?
L24           0 L10 AND ISCHEMIC (W) BRAIN? (W) DISEASE?

=> s l10 and cerebral () thrombosis?
          81793 CEREBRAL
          18393 THROMBOSIS?
            234 CEREBRAL (W) THROMBOSIS?
L25           0 L10 AND CEREBRAL (W) THROMBOSIS?

=> s l10 and cerebral () embolism?
          81793 CEREBRAL
           4541 EMBOLISM?
             91 CEREBRAL (W) EMBOLISM?
L26           0 L10 AND CEREBRAL (W) EMBOLISM?

=> s l10 and transient () ischemic?
         171448 TRANSIENT
          17622 TRANSIENTS
         181175 TRANSIENT
                  (TRANSIENT OR TRANSIENTS)
          37886 ISCHEMIC?
            149 ISCHEM
            149 ISCHEM
                  (ISCHEM)
          37939 ISCHEMIC?
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                (ISCHEMIC? OR ISCHEM)
            459 TRANSIENT (W) ISCHEMIC?
L27           1 L10 AND TRANSIENT (W) ISCHEMIC?

=> s l27 and review/dt
       1738650 REVIEW/DT
L28           0 L27 AND REVIEW/DT

=> s l10 and cerebrovascular? () spasm?
          5949 CEREBROVASCULAR?
         13433 SPASM?
            51 CEREBROVASCULAR? (W) SPASM?
L29           0 L10 AND CEREBROVASCULAR? (W) SPASM?

=> s l10 and thrombosis?
         18393 THROMBOSIS?
L30           8 L10 AND THROMBOSIS?

=> s l30 and review/dt
       1738650 REVIEW/DT
L31           2 L30 AND REVIEW/DT

=> d l31, ibib abs, 1-2

L31  ANSWER 1 OF 2  HCAPLUS  COPYRIGHT 2004 ACS on STN
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Full Text | Citing References

ACCESSION NUMBER: 2002:261513 HCAPLUS
DOCUMENT NUMBER: 137:118914
TITLE: Glycoprotein IIb/IIIa antagonists - from bench to practice
AUTHOR(S): Casserly, I. P.; Topol, E. J.
CORPORATE SOURCE: Department of Cardiovascular Medicine, Cleveland Clinic Foundation, Cleveland, OH, 44195, USA
SOURCE: Cellular and Molecular Life Sciences (2002), 59(3), 478-500
CODEN: CMLSFI; ISSN: 1420-682X
PUBLISHER: Birkhaeuser Verlag
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. The central role played by the αIIbβ3 receptor in platelet aggregation, and hence in platelet **thrombosis**, has led to the development of a no. of parenteral and oral glycoprotein (GP) IIb/IIIa inhibitors for use in cardiovascular disease states, such as acute coronary syndromes and stroke. The predominant effect of these agents is to inhibit platelet aggregation, although studies of αIIbβ3 receptor function and various GP IIb/IIIa inhibitors have demonstrated the potential for these agents to produce effects on other aspects of platelet function, in addn. to non-platelet effects. Overall, clin. studies have demonstrated an impressive beneficial effect for parenteral agents in reducing ischemic complications following percutaneous intervention, and a more modest beneficial effect in the treatment of patients with acute coronary syndromes. Trials with oral GP IIb/IIIa inhibitors in similar patient populations have demonstrated toxicity, manifested by an increased mortality in treated patients. Increased understanding of mol. aspects of both αIIbβ3 receptor function and the effects of GP IIb/IIIa inhibition may help explain some of the inconsistency in recently reported clin. studies with parenteral agents, and the frank toxicity of oral agents. Such studies may also hold the key to the development of newer

agents with enhanced therapeutic benefit.
REFERENCE COUNT: 174 THERE ARE 174 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L31 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2004 ACS on STN
Full Text | Citing References
ACCESSION NUMBER: 2000:54613 HCAPLUS
DOCUMENT NUMBER: 132:73123
TITLE: Glycoprotein IIB/IIIA antagonists: do they have a role in the management of unstable angina?
AUTHOR(S): Redwood, S.; Marber, M.; Jackson, G.
CORPORATE SOURCE: Cardiothoracic Unit, St Thomas' Hospital, London, E1 7EH, UK
SOURCE: International Journal of Clinical Practice (1999), 53(8), 618-622
CODEN: IJCPF9; ISSN: 1368-5031
PUBLISHER: Medicom International
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review with 36 refs. Plaque rupture, platelet aggregation and **thrombosis** have central roles in the pathogenesis of acute coronary syndromes. Despite several trials showing the benefit of aspirin and heparin in patients presenting with unstable angina and acute myocardial infarction, these patients are still at risk. This has prompted the development and evaluation of several new therapeutic agents including low mol. wt. heparin, new antiplatelet drugs (e.g. ticlopidine and clopidogrel), direct thrombin inhibitors, and i.v. and oral glycoprotein IIb/IIIa antagonists. The IIb/IIIa receptor is the "final common pathway" involved in platelet aggregation. Thus, whatever the stimulus for platelet activation, subsequent aggregation is mediated by the IIb/IIIa receptor binding fibrinogen. A variety of antibody, peptide and non-peptide compds. that block the IIb/IIIa receptor have been developed, and several studies have investigated the role of these agents in patients with acute coronary syndromes both within and outside the setting of percutaneous intervention. This article summarizes the studies to date using IIb/IIIa antagonists, and discusses their role in patients with non-ST segment elevation acute coronary syndromes.
REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

Get

```
=> s l10 and arteriosclerosis? () obliteran?
         10325 ARTERIOSCLEROSIS?
           540 OBLITERAN?
           171 ARTERIOSCLEROSIS? (W) OBLITERAN?
L32          0 L10 AND ARTERIOSCLEROSIS? (W) OBLITERAN?

=> s l10 and thromboangiitis?
           198 THROMBOANGIITIS?
L33          0 L10 AND THROMBOANGIITIS?

=> s l10 and burger's () disease?
MISMATCHED QUOTE 'BURGER'S'
Quotation marks (or apostrophes) must be used in pairs,
one before and one after the expression you are setting
off or masking.

=> s l10 and obliteran?
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           540 OBLITERAN?
L34          0 L10 AND OBLITERAN?

=> s l10 and raynaud? () disease?
           668 RAYNAUD?
        785618 DISEASE?
            38 RAYNAUD? (W) DISEASE?
L35          0 L10 AND RAYNAUD? (W) DISEASE?

=> s l10 and burger? () disease?
          5718 BURGER?
        785618 DISEASE?
             4 BURGER? (W) DISEASE?
L36          0 L10 AND BURGER? (W) DISEASE?

=> s l10 and diabetic () angiopathy?
         49865 DIABETIC
          7992 DIABETICS
         53449 DIABETIC
                 (DIABETIC OR DIABETICS)
          1645 ANGIOPATHY?
           855 DIABETIC (W) ANGIOPATHY?
L37          0 L10 AND DIABETIC (W) ANGIOPATHY?

=> s l10 and phlebothrombosis?
            19 PHLEBOTHROMBOSIS?
L38          0 L10 AND PHLEBOTHROMBOSIS?

=> s l10 and vascular () surgery
        124661 VASCULAR
             4 VASCULARS
        124664 VASCULAR
                 (VASCULAR OR VASCULARS)
         40745 SURGERY
           445 SURGERIES
         40998 SURGERY
                 (SURGERY OR SURGERIES)
           275 VASCULAR (W) SURGERY
L39          0 L10 AND VASCULAR (W) SURGERY

=> s l10 and valve () replacement
         52074 VALVE
         28604 VALVES
         66652 VALVE
                 (VALVE OR VALVES)
        105984 REPLACEMENT
          7728 REPLACEMENTS
        111517 REPLACEMENT
                 (REPLACEMENT OR REPLACEMENTS)
           304 VALVE (W) REPLACEMENT
L40          0 L10 AND VALVE (W) REPLACEMENT

=> s l10 and restenosis?
          5654 RESTENOSIS?
L41          6 L10 AND RESTENOSIS?

=> s l41 and review/dt
       1738650 REVIEW/DT
L42          1 L41 AND REVIEW/DT
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=> d 142, ibib abs, 1

L42  ANSWER 1 OF 1  HCAPLUS  COPYRIGHT 2004 ACS on STN
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Full Text | Citing References

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ACCESSION NUMBER:         2003:215934  HCAPLUS
DOCUMENT NUMBER:          138:352321
TITLE:                    Glycoprotein IIb/IIIa inhibition in early
                          intent-to-stent treatment of acute coronary syndromes:
                          EPISTENT, ADMIRAL, CADILLAC, and TARGET
AUTHOR(S):                Moliterno, David J.; Chan, Albert W.
CORPORATE SOURCE:         Department of Cardiovascular Medicine, The Cleveland
                          Clinic Foundation, Cleveland, OH, USA
SOURCE:                   Journal of the American College of Cardiology (2003),
                          41(4, Suppl.), 49S-54S
                          CODEN: JACCDI; ISSN: 0735-1097
PUBLISHER:                Elsevier Science Inc.
DOCUMENT TYPE:            Journal; General Review
LANGUAGE:                 English
AB    A review.  The acute coronary syndromes (ACS), with or without ST-segment
      elevation, share a common pathophysiol. of activated platelets and
      thrombin generation stimulated by plaque erosion and rupture.  Both mech.
      and pharmacol. treatment strategies have evolved in an attempt to improve
      reperfusion at the myocardial tissue level.  Intracoronary stents have
      lowered the incidence of abrupt vessel closure and restenosis, while
      potent platelet inhibition from i.v. glycoprotein IIb/IIIa antagonists has
      reduced the rate of periprocedural myocardial infarction and late
      mortality.  Abciximab has well-established clin. benefits in percutaneous
      revascularization trials, and several recent landmark studies have
      evaluated the efficacy of concomitant abciximab during mech. reperfusion
      therapy in the setting of ACS.  These trials are reviewed, and an overall
      perspective is provided.
REFERENCE COUNT:          56    THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS
                                RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> d his

     (FILE 'HOME' ENTERED AT 11:25:36 ON 01 JUL 2004)

     FILE 'REGISTRY' ENTERED AT 11:25:47 ON 01 JUL 2004
L1             STRUCTURE UPLOADED
L2           0 S L1
L3           0 S L1 FULL
L4             STRUCTURE UPLOADED
L5           0 S L4
L6             STRUCTURE UPLOADED
L7           0 S L6
L8           0 S L6 FULL

     FILE 'HCAPLUS' ENTERED AT 11:35:36 ON 01 JUL 2004
L9           0 S GLYCOPROTEIN IIB/IIIA () ANTAGONIST?
L10         75 S GLYCOPROTEIN? () ANTAGONIST?
L11          2 S L10 AND THROMBUS () FORMATION
L12          1 S L11 AND REVIEW/DT
L13          1 S L10 AND ARTERIAL? () THROMBOSIS?
L14          0 S L13 AND REVIEW/DT
L15          0 S L10 AND ARTERIAL? () SCLEROSIS?
L16          0 S L10 AND ISCHEMIC? () HEART? () DISEASE?
L17          0 S L10 AND ISCHEM? () HEART?
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L18           4 S L10 AND ANGINA? () PECTORIS?
L19           2 S L18 AND REVIEW/DT
L20          10 S L10 AND MYOCARDIAL? () INFARCT?
L21           8 S L20 AND REVIEW/DT
L22           1 S L10 AND CORONARY () THROMBOS?
L23           0 S L22 AND REVIEW/DT
L24           0 S L10 AND ISCHEMIC () BRAIN? () DISEASE?
L25           0 S L10 AND CEREBRAL () THROMBOSIS?
L26           0 S L10 AND CEREBRAL () EMBOLISM?
L27           1 S L10 AND TRANSIENT () ISCHEMIC?
L28           0 S L27 AND REVIEW/DT
L29           0 S L10 AND CEREBROVASCULAR? () SPASM?
L30           8 S L10 AND THROMBOSIS?
L31           2 S L30 AND REVIEW/DT
L32           0 S L10 AND ARTERIOSCLEROSIS? () OBLITERAN?
L33           0 S L10 AND THROMBOANGIITIS?
L34           0 S L10 AND OBLITERAN?
L35           0 S L10 AND RAYNAUD? () DISEASE?
L36           0 S L10 AND BURGER? () DISEASE?
L37           0 S L10 AND DIABETIC () ANGIOPATHY?
L38           0 S L10 AND PHLEBOTHROMBOSIS?
L39           0 S L10 AND VASCULAR () SURGERY
L40           0 S L10 AND VALVE () REPLACEMENT
L41           6 S L10 AND RESTENOSIS?
L42           1 S L41 AND REVIEW/DT

=> s l10 and reocclusion?
            462 REOCCLUSION?
L43           1 L10 AND REOCCLUSION?

=> s l43 and review/dt
        1738650 REVIEW/DT
L44           0 L43 AND REVIEW/DT

=> s l10 and transplantation?
          53093 TRANSPLANTATION?
L45           0 L10 AND TRANSPLANTATION?

=> s l10 and etracorporeal () circulation?
              0 ETRACORPOREAL
         125828 CIRCULATION?
              0 ETRACORPOREAL (W) CIRCULATION?
L46           0 L10 AND ETRACORPOREAL (W) CIRCULATION?

=> s l10 and disseminat? () intravascular? () coagulation?
          17122 DISSEMINAT?
           8769 INTRAVASCULAR?
          96070 COAGULATION?
           1837 DISSEMINAT? (W) INTRAVASCULAR? (W) COAGULATION?
L47           0 L10 AND DISSEMINAT? (W) INTRAVASCULAR? (W) COAGULATION?

=> s l10 and thromb? () thrombocytopenic?
          94675 THROMB?
           1890 THROMBOCYTOPENIC?
            373 THROMB? (W) THROMBOCYTOPENIC?
L48           0 L10 AND THROMB? (W) THROMBOCYTOPENIC?

=> s l10 and thrombocytosis?
            565 THROMBOCYTOSIS?
L49           0 L10 AND THROMBOCYTOSIS?
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=> d his

     (FILE 'HOME' ENTERED AT 11:25:36 ON 01 JUL 2004)

     FILE 'REGISTRY' ENTERED AT 11:25:47 ON 01 JUL 2004
L1             STRUCTURE UPLOADED
L2           0 S L1
L3           0 S L1 FULL
L4             STRUCTURE UPLOADED
L5           0 S L4
L6             STRUCTURE UPLOADED
L7           0 S L6
L8           0 S L6 FULL

     FILE 'HCAPLUS' ENTERED AT 11:35:36 ON 01 JUL 2004
L9           0 S GLYCOPROTEIN IIB/IIIA () ANTAGONIST?
L10         75 S GLYCOPROTEIN? () ANTAGONIST?
L11          2 S L10 AND THROMBUS () FORMATION
L12          1 S L11 AND REVIEW/DT
L13          1 S L10 AND ARTERIAL? () THROMBOSIS?
L14          0 S L13 AND REVIEW/DT
L15          0 S L10 AND ARTERIAL? () SCLEROSIS?
L16          0 S L10 AND ISCHEMIC? () HEART? () DISEASE?
L17          0 S L10 AND ISCHEM? () HEART?
L18          4 S L10 AND ANGINA? () PECTORIS?
L19          2 S L18 AND REVIEW/DT
L20         10 S L10 AND MYOCARDIAL? () INFARCT?
L21          8 S L20 AND REVIEW/DT
L22          1 S L10 AND CORONARY () THROMBOS?
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L24          0 S L10 AND ISCHEMIC () BRAIN? () DISEASE?
L25          0 S L10 AND CEREBRAL () THROMBOSIS?
L26          0 S L10 AND CEREBRAL () EMBOLISM?
L27          1 S L10 AND TRANSIENT () ISCHEMIC?
L28          0 S L27 AND REVIEW/DT
L29          0 S L10 AND CEREBROVASCULAR? () SPASM?
L30          8 S L10 AND THROMBOSIS?
L31          2 S L30 AND REVIEW/DT
L32          0 S L10 AND ARTERIOSCLEROSIS? () OBLITERAN?
L33          0 S L10 AND THROMBOANGIITIS?
L34          0 S L10 AND OBLITERAN?
L35          0 S L10 AND RAYNAUD? () DISEASE?
L36          0 S L10 AND BURGER? () DISEASE?
L37          0 S L10 AND DIABETIC () ANGIOPATHY?
L38          0 S L10 AND PHLEBOTHROMBOSIS?
L39          0 S L10 AND VASCULAR () SURGERY
L40          0 S L10 AND VALVE () REPLACEMENT
L41          6 S L10 AND RESTENOSIS?
L42          1 S L41 AND REVIEW/DT
L43          1 S L10 AND REOCCLUSION?
L44          0 S L43 AND REVIEW/DT
L45          0 S L10 AND TRANSPLANTATION?
L46          0 S L10 AND ETRACORPOREAL () CIRCULATION?
L47          0 S L10 AND DISSEMINAT? () INTRAVASCULAR? () COAGULATION?
L48          0 S L10 AND THROMB? () THROMBOCYTOPENIC?
L49          0 S L10 AND THROMBOCYTOSIS?

=> s inhibitor? () aggregation?
        883196 INHIBITOR?
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         92759 AGGREGATION?
L50         19 INHIBITOR? (W) AGGREGATION?

=> s l50 and thrombus? () formation?
          7247 THROMBUS?
       2501180 FORMATION?
          3029 THROMBUS? (W) FORMATION?
L51          0 L50 AND THROMBUS? (W) FORMATION?

=> s l50 and thromb?
         94675 THROMB?
L52          4 L50 AND THROMB?

=> s l52 and review/dt
       1738650 REVIEW/DT
L53          0 L52 AND REVIEW/DT

=> s l50 and arterial? () sclerosis?
         79957 ARTERIAL?
         18569 SCLEROSIS?
            36 ARTERIAL? (W) SCLEROSIS?
L54          0 L50 AND ARTERIAL? (W) SCLEROSIS?

=> s l50 and ischemic? () heart?
         37886 ISCHEMIC?
           149 ISCHEM
           149 ISCHEM
                 (ISCHEM)
         37939 ISCHEMIC?
                 (ISCHEMIC? OR ISCHEM)
        312570 HEART?
          5475 ISCHEMIC? (W) HEART?
L55          0 L50 AND ISCHEMIC? (W) HEART?

=> s l50 and angina? () pectoris?
          7629 ANGINA?
          5768 PECTORIS?
          5607 ANGINA? (W) PECTORIS?
L56          0 L50 AND ANGINA? (W) PECTORIS?

=> s l50 and myocardial? () infarction?
         54567 MYOCARDIAL?
         26585 INFARCTION?
         16658 MYOCARDIAL? (W) INFARCTION?
L57          0 L50 AND MYOCARDIAL? (W) INFARCTION?

=> s l50 and cerebral () thrombos?
         81793 CEREBRAL
         21416 THROMBOS?
           240 CEREBRAL (W) THROMBOS?
L58
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